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Reverse Redistribution of Thallium-201: A Sign of Nontransmural Myocardial Infarction With Patency of the Infarct-Related Coronary Artery

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The pattern of reverse redistribution on the day 10 poststreptokinase resting thallium-201 myocardial scintigrams is a common finding in patients who have undergone streptokinase therapy in evolving myocardial infarction. To investigate this phenomenon, 67 patients who underwent streptokinase therapy were studied pre- and 10 days poststreptokinase therapy resting thallium-201 studies, poststreptokinase therapy resting radionuclide ventriculography and coronary arteriography (60 of the 67 patients). Of the 67 patients, 50 (75%) showed the reverse redistribution pattern on the day 10 thallium-201 study (Group I), 9 (13%) had a nonreversible defect (Group II) and the remaining 8 (12%) had a normal study or showed a reversible defect (Group III). The reverse redistribution pattern was associated with patency of the infarct-related artery (100%), quantitative improvement in resting thallium-201 defect size from day 1 to day 10 study (94%) and normal or near normal wall motion on day 10 radionuclide ventriculography (80% of segments with marked and 54% of those with mild reverse redistribution). In contrast, nonreversible defects were associated with significantly less frequent patency of the infarct-related artery (67%, $p = 0.01$), improvement in defect size (11%, $p < 0.001$) and normal or near normal wall motion (21%, $p < 0.05$). Group III patients were similar to Group I with respect to these variables. The quantitated thallium-201 percent washout was higher in the regions with the reverse redistribution pattern (49 ± 15%) compared with the contralateral normal zone (24 ± 15%, $p < 0.001$).

The findings indicate that the reverse redistribution pattern results from a higher than normal washout rate of thallium-201 and suggest that this pattern is a sign of nontransmural myocardial infarction with a patent infarct-related coronary artery.

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The characteristic pattern of thallium-201 scintigrams in myocardial ischemia is a perfusion defect noted on early postinjection images, either at rest or during stress, that normalizes after a time delay of several hours ("reversible defect") (1–6). In contrast, a region with myocardial infarction typically shows a perfusion defect that remains unchanged ("persistent defect") (1,7). Infrequently, however, a perfusion defect appears or becomes more evident on the delayed than on the early image. This so-called pattern of "reverse redistribution" on stress redistribution thallium-201 images has been previously reported (8,9), but its etiology, significance and clinical implications are not well understood. In patients with evolving myocardial infarction who have undergone early streptokinase therapy, we have frequently observed the "reverse redistribution" pattern on their day 10 poststreptokinase resting thallium-201 study (Fig. 1).

The aims of this study were 1) to assess the frequency of this thallium-201 reverse redistribution pattern in patients with an acute myocardial infarction who received streptokinase therapy, and 2) to evaluate its clinical significance with respect to patency of the infarct-related coronary artery and the presence of viable myocardium in the reperfused

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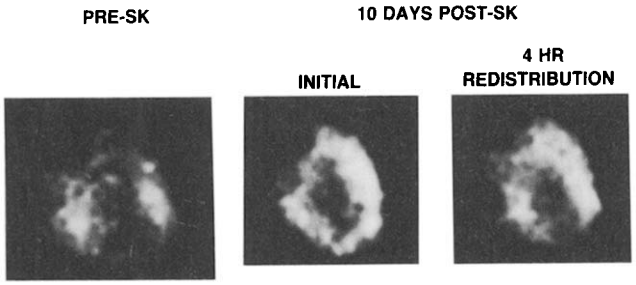

Figure 1. Anterior view thallium-201 images prestreptokinase (SK) and 10 days poststreptokinase coronary thrombolysis in a patient presenting with evolving inferior myocardial infarction. The prestreptokinase image demonstrates extensive apical and inferior thallium defects. The initial resting image on day 10 is normal; however, the 4 hour redistribution image demonstrates the pattern of reverse redistribution in the apical and distal inferior regions.

region as suggested by regional wall motion and poststreptokinase therapy decrease in thallium-201 defect size.

Methods

Study patients. The patient population consisted of 67 consecutive patients who satisfied the following inclusion criteria: 1) they were hospitalized within 3 hours of onset of persistent chest pain unresponsive to nitroglycerin, 2) they showed electrocardiographic evidence of transmural ischemia (at least 1 mm ST segment elevation in more than one lead), 3) they underwent streptokinase therapy, 4) they had resting thallium-201 studies before the intervention, and 5) they underwent resting thallium-201 and radionuclide ventriculographic studies 7 to 10 days after the intervention. There were 53 men and 14 women with a mean age of 60 years (range 32 to 87). The admission electrocardiogram showed evolving infarction of the anterior wall in 28 patients and of the inferior wall in 39 patients. Six patients had documented prior myocardial infarction.

Protocol for thrombolytic therapy with streptokinase. In 15 patients who were admitted before August 1982, streptokinase was administered by way of the intracoronary route according to the method previously described by our group (10). In patients undergoing intracoronary streptokinase therapy, reperfusion was assessed by repeat intracoronary contrast injection in the acute setting. In the remaining 52 patients who were admitted after August 1982, streptokinase was administered intravenously in a dose of 750,000 U over 15 to 20 minutes. In 17 of the 52 patients, a repeat dose of 750,000 U was administered after 20 minutes. In patients undergoing intravenous streptokinase infusion, reperfusion was judged by termination of ischemia as evidenced by the resolution of ST segment elevation and abatement of chest pain, as well as early rise and peaking of frequently sampled serum levels of creatine kinase (CK) (11). The status of the infarct-related coronary artery was documented by coronary angiography in 45 of the 52 patients 3 to 5 days after the therapy as described later.

The mean time (and its range) from onset of chest pain to reperfusion was 221 ± 75 minutes (range 123 to 334) for the intracoronary and 173 ± 70 minutes (range 51 to 345) for the intravenous approach. There was evidence for CK-MB release in all patients which ranged from 6 to 291 IU (average 126 ± 88 [SD]). After streptokinase infusion, all 67 patients received anticoagulation therapy with continuous intravenous infusion of heparin to maintain a partial thromboplastin time of greater than 100 seconds.

Coronary angiography. Coronary arteriograms in multiple views were obtained in 60 of the 67 patients. In all of the 15 patients undergoing intracoronary streptokinase therapy, coronary arteriography was performed on admission. In 45 of the 52 patients undergoing intravenous streptokinase administration, coronary arteriography was performed 3 to 5 days after the therapy. The coronary angiograms were reviewed by two blinded experienced angiographers for presence or absence of patency in the infarct-related coronary artery. In the 15 patients undergoing intracoronary streptokinase administration, the artery of infarction was identified by direct visualization of the occluded coronary artery. The artery of infarction was apparent in 27 of the 45 patients receiving intravenous streptokinase who had anterior myocardial infarction. In the remaining 38 of the 45 patients with inferior myocardial infarction, the artery of the infarction was identified by 1) angiographic criteria of an ulcerated atheromatous plaque, that is, indistinct luminal margins or subintimal ulceration of the coronary artery, and 2) by the regional pattern of prestreptokinase acute electrocardiographic changes, left ventricular dysfunction (on contrast left ventriculography) and perfusion (on the prestreptokinase thallium-201 study).

Thallium-201 scintigraphy. All patients received an intravenous thallium-201 dose of 2 mCi on admission before streptokinase administration followed by planar thallium-201 imaging. In the intracoronary streptokinase group, different brief acquisition views were inspected and the view with the largest perfusion defect was imaged for 10 minutes. In the intravenous streptokinase group, sequential planar images were obtained in the anterior, 45° left anterior oblique and steep left anterior oblique views for 10 minutes per view; the view with the largest perfusion defect was chosen for visual and quantitative analysis. In a few patients, imaging was not started until streptokinase infusion was initiated. Despite this delay, the imaged thallium-201 defect size closely represented the size of the initially jeopardized myocardium at prereperfusion state because 1) thallium-201 was injected before streptokinase administration, and 2) with some delay in imaging, no significant change of thallium-201 pattern is expected to occur in this setting (12).

Seven to 10 days after streptokinase therapy, all 67 patients received a repeat 2 mCi dose of thallium-201 intravenously. Sequential images were then obtained 10 minutes (initial) and 4 hours (delayed) after thallium-201 injection, duplicating the viewing angles used for the prestreptokinase study. For imaging, a mobile scintillation camera (Ohio Nuclear, Series 410) equipped with an all-purpose collimator was used. Images were recorded by computer on a 128 × 128 × 8 bit matrix.

Assessment of thallium-201 reverse redistribution and other redistribution patterns. The pattern of redistribution was evaluated on the day 10 poststreptokinase thallium-201 study by visually comparing the initial with the delayed image. Images were visually scored by three experienced observers blinded to the clinical findings and the results of other tests. The observers divided each view into five segments and scored each segment by consensus using a semiquantitative four point scoring system in which 0 = normal uptake, 1 = mildly decreased uptake, 2 = moderately decreased uptake and 3 = severely decreased myocardial uptake. Although a previously described quantitative thallium-201 image analysis (13) has been useful for the detection (14) and sizing (15) of thallium-201 perfusion defects, it has not been validated for the assessment of reversibility of a given perfusion defect. Therefore, in this and another study (16), we have used a semiquantitative visual scoring system for assessment of reversibility. Visual assessment of thallium-201 uptake in 345 myocardial segments by two expert observers (J.M. and D.B.) in our laboratory has shown an interobserver agreement of 83% (287/345) for identical grading of the severity of thallium-201 perfusion defects and 88% (305/345) for identical grading of the degree of change from initial to delayed images.

Reverse redistribution was considered to be present if a segmental perfusion deteriorated by a score of at least one. The magnitude of reverse redistribution was classified into three types: type a (marked reverse redistribution) defined as initial to delayed scores of 0-2, 0-3 and 1-3; type b (mild reverse redistribution) with scores of 0-1 and 1-2 and type c (minimal reverse redistribution) with a score of 2-3. Nonreversible segments were defined as those with scores of 1-1, 2-2 or 3-3. Segments were considered normal with the score of 0-0 and were considered reversible with scores of 3-0, 3-1, 3-2, 2-0, 2-1 and 1-0.

Assessment of thallium-201 defect size. Thallium-201 defect size on both pre- and 10 day poststreptokinase studies was assessed visually and quantitatively. In the six patients with old myocardial infarction, the area of prior infarction (identified by the electrocardiogram) was excluded from the defect analysis. Visual defect size was represented by summing visual scores of all segments subtended by the infarct-related coronary artery. Thallium-201 defect size was also quantitated using a circumferential profile analysis technique similar to that previously described by our group (13) for the analysis of postexercise images. The patient's circumferential profile was compared with the previously established normal limit circumferential profile and the thallium-201 defect size was defined as the size of the area lying between the normal limit profile and the portion of the patient's profile falling below the normal limit profile. The area (quantitative defect size) was expressed numerically in units that ranged from 0 (no defect) to a maximum of 1,800 (largest defect observed). A decrease of less than 50 U in the quantitative defect size from the pre- to 10 day poststreptokinase study represented improvement. The cutoff of 50 U was based on our intraobserver variability study for the quantitative analysis, demonstrating that a change in the quantitative perfusion defect score of less than 50 U is within the variability of the quantitative method.

Measurement of percent regional washout. In the last 23 of the 50 patients with reverse redistribution, the myocardial thallium-201 percent washout profiles on the day 10 study were determined using a previously described algorithm (13). The mean percent washout of the region demonstrating reverse redistribution was compared with that of the contralateral normal zone.

Radionuclide ventriculography. Resting radionuclide ventriculograms were obtained shortly after the day 10 thallium-201 study. Each patient received an intravenous injection of 25 mCi of technetium-99m–labeled autologous red blood cells, and multiple-gated acquisition was used (200,000 counts per frame, 20 frames/cycle) to obtain images in the same three views as utilized for thallium-201 imaging. Regional wall motion was visually assessed by three experienced observers blinded to all other data. The left ventricle was divided in each view into five segments and wall motion was scored using a five point scoring system (17) in which 3 = normal motion, 2 = mild hypokinesia (near normal), 1 = severe hypokinesia, 0 = akinesia and −1 = dyskinesia.

Statistical analysis. Fisher's exact test was used to compare the proportion of patients in Groups I, II and III who demonstrated a patent infarct-related coronary artery and improvement in thallium-201 defect size. Fisher's exact test was also used to compare the frequency of normal and near normal wall motion among segments with different types (a, b, c) of reverse redistribution, nonreversible segments and the segments with normal/reversible defects. The analysis of variance was used to compare the mean improvement in defect size between Groups I and II and between Groups II and III. The unpaired *t* test was used to compare the mean percent washout between the regions with reverse redistribution and the normal zones. The test of variance for repeated measurements was used to assess sequential changes in visual thallium-201 defect score from prestreptokinase study to the day 10 poststreptokinase initial and redistribution studies. A probability (p) value of less than 0.05 represented a significant difference.

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Results

Frequency of reverse redistribution pattern compared with other patterns of thallium-201 redistribution. Based on the pattern of thallium-201 redistribution on the day 10 study, patients were divided into three groups. Fifty (75%) of the 67 patients demonstrated the reverse redistribution pattern in at least one myocardial segment (Group I). In this group, the magnitude of change in visual segmental score was 1 in 37 of the 50 patients and 2 in 13 patients. In all 50 patients, the reverse redistribution pattern occurred solely in the reperfused myocardium as evidenced by the location of the initially jeopardized myocardium on the prestreptokinase study. The involved region was entirely nonreversible in 9 (13%) of the 16 patients (Group II). In the remaining 8 patients (12%) the reperfused myocardium was either normal or demonstrated reversible defects (Group III).

The initially jeopardized myocardium of the 67 patients consisted of 293 segments. Of these, 134 segments showed the pattern of reverse redistribution, 99 segments were nonreversible and 60 segments were normal or showed reversible defects (normal/reversible). Thus, reverse redistribution was noted in 75% (50 of 67) of patients and 45% (134 of 293) of segments. Of the 134 segments with reverse redistribution, 20 had a type a, 96 had a type b and 18 had a type c reverse redistribution pattern.

Coronary arteriographic correlates of different patterns of redistribution. Patency of the infarct-related coronary artery was evaluated in the 60 of the 67 patients who were catheterized (47 of 50 in Group I, 6 of 9 in Group II and 7 of 8 in Group III). In all 47 catheterized patients in Group I, the infarct-related coronary artery was patent, whereas of the 6 catheterized Group II patients, 4 (67%) demonstrated a patent infarct-related coronary artery ($p = 0.01$). Group III catheterized patients were similar to Group I in that all seven patients had an angiographically demonstrated patent infarct-related coronary artery. Stated differently, neither of the two patients with a persistently occluded infarct-related artery demonstrated the reverse redistribution pattern. Instead, both of these patients showed a nonreversible thallium-201 perfusion defect. Fourteen (30%) of the 47 patients with a patent infarct-related coronary artery and reverse redistribution pattern had subtotal residual coronary stenosis.

Relation between different thallium-201 redistribution patterns and pre- to 10 day poststreptokinase improvement in perfusion defect size (Fig. 2). The quantitative improvement in perfusion defect size (by comparison of day 1 and day 10 thallium-201 resting scintigrams) was greater in Group I (425 ± 266 units) than in Group II (27 ± 57 units) ($p < 0.001$). Definite improvement (poststreptokinase decrease in perfusion defect >50 U) was present in 47 (94%) of the 50 Group I patients and in only 1 (11%) of the 9 Group II patients ($p < 0.001$). The improvement in perfusion defect size in Group III was 210 ± 190 U, also significantly greater than that of Group II ($p < 0.01$).

Sequential changes in thallium-201 defect size in patients with the reverse redistribution pattern. In the 50 patients with a reverse redistribution pattern, the total visual thallium-201 defect size score on the prestreptokinase study significantly decreased from 12.1 ± 6.9 (mean ± SD) to 5.5 ± 5.1 on the day 10 initial study ($p < 0.01$) and increased to 8.4 ± 5.4 ($p < 0.001$) on the day 10 redistribution study. Thus, despite the pattern of reverse redistribution, the thallium-201 defect score on the day 10 redistribution study (8.4 ± 5.4) was significantly lower ($p < 0.01$) than that observed on the prestreptokinase study (12.1 ± 6.9).

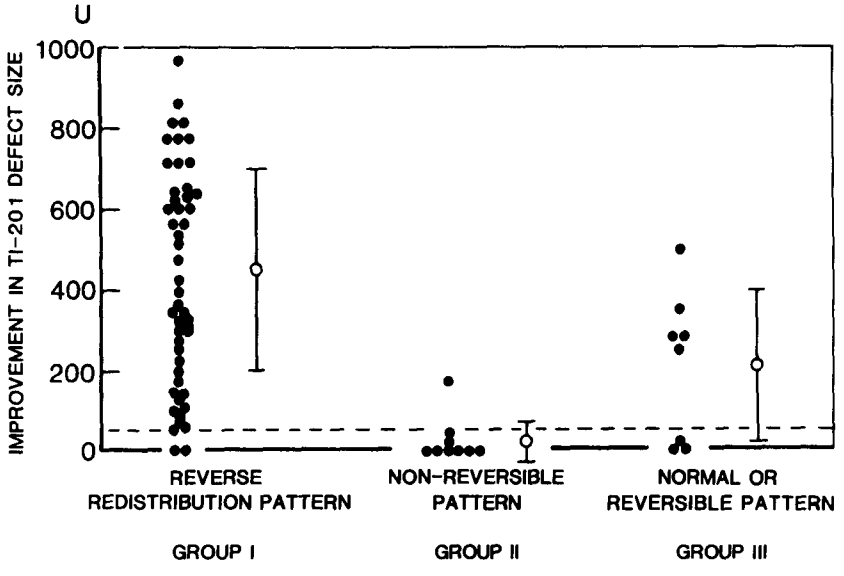


Figure 2. Relation between the improvement in the quantitated thallium-201 (Tl-201) defect size (from pre- to day 10 poststreptokinase therapy) and the pattern of thallium-201 redistribution on the day 10 study.

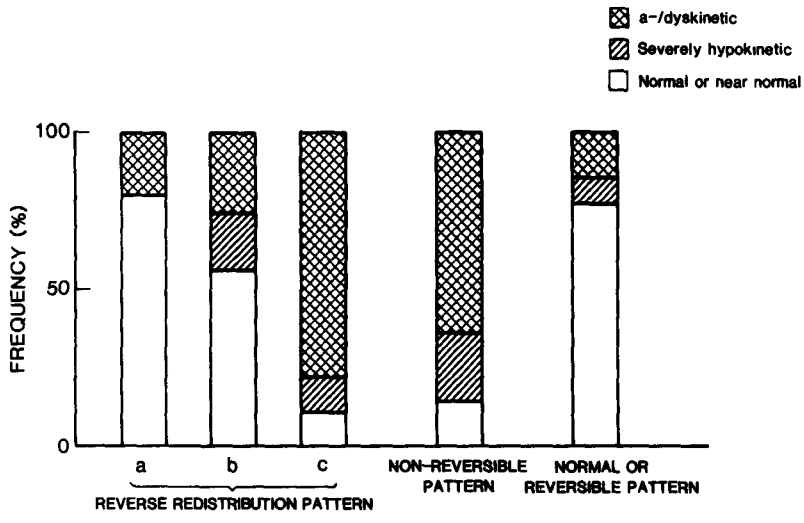


Figure 3. Segmental relation between different thallium-201 (Tl-201) redistribution patterns and regional wall motion; a, b and c refer to different types of reverse redistribution. Type a = marked reverse distribution; type b = mild reverse distribution and type c = minimal reverse distribution.

Segmental relation between different thallium-201 redistribution patterns and regional wall motion (Fig. 3). In each of the 293 segments, the pattern of thallium-201 redistribution and corresponding regional wall motion were compared. Regarding segments with reverse redistribution, 80% (16 of 20) of type a segments and 54% (52 of 96) of type b segments had normal or near normal wall motion. In contrast, only 11% (2 of 18) of type c segments had normal or near normal wall motion. Nonreversible segments showed a significantly lower ($p < 0.05$) frequency of normal or near normal wall motion (21 of 99, 21%) compared with type a and type b of the reverse redistribution group. These nonreversible segments, however, were similar to type c (21 versus 11%, p = NS). Normal or reversible segments were similar to type a in that 77% (44 of 60) had normal or near normal wall motion.

Akinetic or dyskinetic wall motion was frequent in type c reverse redistribution and nonreversible segments (78 and 65%, respectively) and less common ($p < 0.05$) in type a, type b and the normal/reversible segments (22, 26 and 15%, respectively).

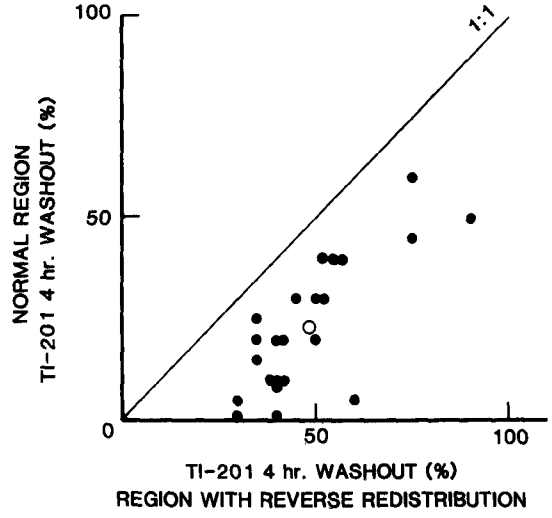
Thallium-201 washout rate in regions with reverse redistribution pattern compared with the noninvolved region. In the 23 of the 50 patients with reverse redistribution in whom the percent washout of thallium-201 from the initial to the 4 hour redistribution day 10 study was quantified, the thallium-201 washout was significantly faster in the region with the reverse redistribution pattern than in the zone supplied by arteries other than the artery of infarction (49 ± 15 versus 24 ± 15%, $p < 0.001$) (Fig. 4). This inequality of regional percent washout of thallium-201 washout explains the occurrence of reverse redistribution pattern on visual analysis.

Follow-up nuclear stress studies. Twenty-four of the 67 patients underwent follow-up stress redistribution thallium-201 scintigraphy at a mean of 8 weeks (range 2 to 30) after streptokinase therapy. Of the 24 patients, 19 showed the reverse redistribution pattern on their day 10 resting study; 16 (84%) of these 19 patients also had the pattern of reverse redistribution on their follow-up stress redistribution study. The remaining five patients who did not show reverse redistribution on the day 10 study demonstrated this pattern on their follow-up study.

Discussion

The pattern of "reverse redistribution" on stress redistribution thallium-201 images has been previously reported (8,9). Although it is generally accepted that the myocardial

Figure 4. Faster 4 hour percent washout of thallium-201 (Tl-201) in the region demonstrating reverse redistribution compared with that of the contralateral normal region. If the washout rates were equal in these zones, the points would cluster around the illustrated **line of identity**. The **open circle** represents the mean percent washout in the two zones.



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segment manifesting thallium-201 reverse redistribution has a relatively higher thallium-201 washout rate than that of the adjacent regions, the mechanism for such difference is controversial. Hecht et al. (9) demonstrated that reverse redistribution occurred more in regions supplied by a stenosed coronary artery. Tanasescu et al. (8) showed that this phenomenon, termed "apparent worsening," occurred in myocardial segments that were perfused by a normal or the least stenosed coronary artery.

Reverse redistribution after streptokinase therapy for myocardial infarction. Our findings indicate that in patients who have undergone streptokinase therapy during the acute phase of their myocardial infarction, the pattern of reverse redistribution on the day 10 resting redistribution thallium-201 study is observed in 75% of studies and 45% of myocardial segments. Our results in the patients undergoing coronary angiography also demonstrated that all the myocardial regions with the reverse redistribution pattern were subtended by a patent coronary artery. Furthermore, reverse redistribution was not seen in either of the two patients with a persistently occluded infarct-related artery.

The pattern of reverse redistribution appears to be also related to the presence of both necrotic and viable myocardium in the reperfused zone. In this study, an element of necrosis in the initially jeopardized zone was evidenced by release of CK-MB in all patients. Presence of viable myocardium was suggested by improvement in the resting thallium-201 defect from the pre- to 10 day poststreptokinase study, observed in 94% of the patients with reverse redistribution. It could be argued that this improvement in thallium-201 defect size merely represented reestablishment of coronary blood flow to infarcted myocardium in the initially jeopardized zone. However, the improvement in thallium-201 defect size (as assessed by changes in total visual defect size) was significant not only between the prestreptokinase study and the day 10 initial study, but also between the prestreptokinase study and the day 10 redistribution study. Further evidence for the presence of viable tissue in the reperfused region was provided by the day 10 resting wall motion study that showed normal or near normal wall motion in 80% of type a segments, which had marked reverse redistribution (two score worsening from initial imaging to redistribution), and in 54% of type b segments with mild reverse redistribution. In contrast, normal or near normal wall motion was observed in only 21% of segments with nonreversible defects (type c). These associations suggest that the pattern of reverse redistribution reflects reestablished blood flow to a myocardial region with both viable and necrotic tissues.

Our finding of a combination of necrotic and viable tissue in the reperfused zone is not unexpected. Several studies (10,15,18–20) have shown that streptokinase thrombolysis in the early phase of acute myocardial infarction results in a variable amount of myocardial salvage. The extent of necrosis depends on total ischemic time (16) as well as unknown variables that influence the rate of progression of necrosis in an individual patient. Myocardial necrosis almost always starts from the subendocardium. After the early reperfusion, the subepicardial layers of the myocardium are the regions with most extensive salvage (18).

Mechanisms of reverse redistribution pattern. Our results demonstrate that the phenomenon of reverse redistribution is caused by more rapid washout of thallium-201 in the involved segments than in myocardial regions supplied by arteries other than the artery of infarction. The mechanisms underlying this phenomenon in our patients remain speculative but the following might be considered:

1) Higher than normal blood flow to the noninfarcted tissue in the reperfused zone could explain both the near normal day 10 initial thallium-201 pattern and the subsequent rapid thallium-201 washout observed in our patients. Because the initial distribution of thallium-201 is related to regional blood flow (20), higher than normal flow to the noninfarcted tissue in the reperfused zone would result in higher than normal concentration of thallium-201 in this region, offsetting the significantly decreased thallium-201 uptake in the necrotic portion of the reperfused myocardium. Moreover, the regional washout rate of thallium-201 is related to the initial thallium-201 concentration (and to regional coronary flow) (21,22). Thus, the washout rate of reperfused myocardium with higher than normal flow to the noninfarcted tissue would be higher than that of the contralateral normal myocardium. This rapid washout rate would lead to worsening of a defect from the initial to the 4 hour postinjection image, and thus the pattern of reverse redistribution.

2) Normal blood flow to the noninfarcted tissue in the reperfused zone with decreased blood flow in the infarcted portion of the reperfused zone associated with resting hypoperfusion of the contralateral myocardial region could also explain the pattern of reverse redistribution. With this mechanism, both the reperfused zone and the contralateral region would have decreased absolute concentrations of thallium-201 on the initial image but no relative defect would be present. Subsequently, normal thallium-201 washout from the reperfused zone combined with slow thallium-201 washout from the contralateral ischemic zone would result in the pattern of reverse redistribution. This mechanism appears to be an unlikely explanation for the findings in our patients because the washout rate of thallium-201 from the contralateral segments was similar to that normally observed (23).

3) Significant thallium-201 uptake by the necrotic tissue or the interstitial compartment in the reperfused zone, or both, could also be postulated to explain reverse redistribution of thallium-201 in our patients (24). This mechanism, combined with normal thallium-201 uptake by the reperfused viable myocardium (with normal blood flow), would result in an overall thallium-201 uptake in the reperfused zone that would appear normal or near normal. Because the washout rate of thallium-201 from the interstitial compart-

ment or the necrotic tissue might be faster than the normal myocardium, the overall thallium-201 washout rate of the reperfused zone would be faster than that of the contralateral normal myocardium, resulting in the pattern of reverse redistribution. Our findings, however, suggest that this alternative explanation is not the predominant factor in the pattern of reverse redistribution. If this were the case, one would not expect the association of normal or near normal wall motion with the pattern of reverse redistribution, which was common in our patients. Furthermore, if interstitial thallium-201 uptake in recently infarcted myocardium were responsible for the pattern, one would not expect this pattern to be a persistent phenomenon. The fact that 16 of 19 of our patients undergoing delayed stress redistribution thallium-201 scintigraphy demonstrated this pattern in the delayed state suggests that interstitial thallium-201 uptake in persistent edema postinfarction is an unlikely mechanism for this process.

The findings in this paper describe a subset of patients with evidence of myocardial infarction undergoing streptokinase therapy. Thallium-201 reverse redistribution may also occur in patients without infarction who have been found to have no objective evidence of cardiac disease. In these patients, the finding may represent inhomogeneous washout of thallium-201 as a normal variant. Furthermore, the frequency of the reverse redistribution pattern in patients with acute myocardial infarction not receiving streptokinase infusion is not known.

Implications. Thallium-201 reverse redistribution in patients after streptokinase therapy is caused by faster than normal thallium-201 washout in the reperfused region and is a sign of successful reopening of the occluded coronary artery and presence of viable myocardium in the reperfused zone. Confirmation of the association with viable myocardium is provided by the common occurrence of normal or near normal wall motion in the segments demonstrating significant reverse redistribution. A possible implication of these findings is that in patients with a previous myocardial infarction, reverse redistribution of thallium-201 suggests that the infarction is nontransmural and that the region is perfused by the artery of infarction that is patent, by an open graft or by collateral circulation.

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